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# Efficacy of intravenous lipid emulsion (ILE) in the treatment of acute aluminum phosphide toxicity at Alexandria Main University Hospital, Egypt

The efficacy of intravenous lipid emulsion in acute aluminium phosphide toxicity

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## Abstract

Aim: Aluminum phosphide (AIP) poisoning causes high mortality. The present study evaluated the efficacy of SMOFlipid in acute unstable cases of aluminum phosphide poisoning in the Intensive Care Unit of Alexandria Main University Hospital, Egypt.

Material and Methods: The study included 66 patients, divided into a case group who received SMOFlipid, as well as standard hemodynamic stabilization measures. They were collected prospectively during a ten-month period. The control group received the standard measures. Data of these patients were collected retrospectively from the patients' files of the intensive care unit of Alexandria Main University Hospital during a one-year period.

Results: There was a significant decrease in the mortality rate in the case group (61.3%) compared with the control group (94.3%). Systolic and diastolic blood pressure showed significant increases after SMOFlipid infusion (p=0.019 and 0.017, respectively) compared with controls (p=0.006 for both variables). There was a significant increase in the level of bicarbonate after infusion of SMOFlipid compared to pre-infusion levels (p=0.006). Significant increases in the levels of liver enzymes, triglycerides, and bilirubin were observed after infusion of SMOFlipid compared to pre-SMOF levels (p= <0.001).

Discussion: SMOFlipid caused a significant decrease in mortality rate of unstable acute ALP poisoning. Hemodynamic as well as acid-base status improved after infusion.

## Keywords

Aluminum Phosphide, Unstable Cases, Acute Toxicity, Intravenous Lipid Emulsion, SMOFlipid

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# Introduction

Aluminium phosphide (AIP) is a toxic compound that causes high mortality after acute exposure via different routes. It is a kind of pesticide that is used to protect grains stored in stores [1]. As it is extremely toxic, easily accessible and low in price, it is considered a dangerous source of severe toxicity in developing countries. Exposure to AIP is mostly suicidal, unusually accidental and infrequently homicidal [2].

Each tablet of AIP contains 56% aluminium phosphide and 44% ammonium carbonate that is included in the tablet to avoid self-detonation of phosphine gas (PH3) released upon the exposure of aluminum phosphide to moisture. This lipid-soluble phosphine gas is the active constituent of the AIP tablet, which leads to oxidative stress with the formation of free radicals and inhibition of cytochrome oxidase [3-5].

Intravenous lipid emulsion (ILE) is a combination of long-chain fatty acids that has been utilized to offer essential fatty acids for cases that need parenteral nutrition (PN). At the same time, it is considered a solid basis of calories to decrease the required amount for PN [6,7].

However, ILE has non-nutritional applications as well. It can be used as a treatment for poisonings involving toxic agents that have lipid-soluble properties. Administration of intravenous (IV) lipid emulsion can help in the recovery of patients with cardiovascular collapse [8].

SMOFlipid is a white, homogenous, sterile, non-pyrogenic lipid emulsion for intravenous infusion. The lipid content of SMOFlipid is 0.20 g/ml and involves a combination of fish oil, medium-chain triglycerides (MCTs), soybean oil and olive oil. The mean essential fatty acid content in SMOFlipid is 35 mg/ml (range 28 to 50 mg/ml) linoleic acid (Omega-6) and 4.5 mg/ml (range of 3 to 7 mg/ml)  $\alpha$ -linoleic acid (Omega-3). The phosphate content is 15 mmol/l [9].

The fat emulsion generates a lipid sink, which attracts the toxic agent from the tissues into the lipid phase and separates it from the aqueous plasma, where it cannot produce its pharmacologic action [10].

However, existing endorsements for the use of lipid rescue as a treatment for lipid-soluble toxic agents have been obtained only from case reports and animal studies; there are no available data from wide-scale studies. Therefore, the present study aimed to evaluate the efficacy of intravenous lipid emulsion as a line of treatment for acute unstable cases of aluminium phosphide poisoning admitted to the Intensive Care Unit of Alexandria Main University Hospital, Egypt.

# Material and Methods

# Patients

The present study was conducted on 66 patients. They were divided into two groups: the case group and the control group. The case group received SMOFlipid according to the regimen described in detail in the methods section, in addition to the standard treatment presented for such cases. They were collected prospectively during ten months, from September 1, 2019 to the end of February 2020. The period from early March 2020 to late September 2020 was skipped due to the COVID-19 pandemic. Then, from the beginning of October 2020 to the end of January 2021, the cases were collected again.

During this period, 31 patients with acute AIP toxicity were hemodynamically unstable and admitted to the Intensive Care Unit of Alexandria Main University Hospital.

The control group included 35 patients who received the standard treatment for hemodynamically unstable AIP toxicity cases in the Intensive Care Unit. The data of these patients were retrospectively collected from the patients' files of the Intensive Care Unit of Alexandria Main University Hospital from early September 2018 to the end of August 2019.

The current study included patients who received appropriate, high-quality supportive care, those for whom no further conventional therapy options were available, and patients who continued to be hemodynamically unstable or were in cardiopulmonary arrest (grade 3 of the poisoning severity score).

Patients with a history of hypersensitivity to fish, egg, soya or peanut protein or to any of the active ingredients or excipients, severe hyperlipidemia, and severe liver insufficiency were excluded from the study. In addition, the present study excluded cases with severe blood coagulation disorders, severe renal insufficiency without access to hemofiltration or dialysis, and general contraindications to infusion therapy, such as acute pulmonary edema, hypervolemia, and decompensated cardiac insufficiency.

# Methods

In the current work, the patients in the control group received standard measures for such cases, depending on hemodynamic stabilization. These measures were as follows:

- Airway protection

- Breathing monitoring and endotracheal tube insertion and mechanical ventilation, if indicated

- Circulatory support: intravenous fluid administration, vasoactive drug infusion

- Sodium bicarbonate infusion to buffer metabolic acidosis

- Proton pump inhibitor to guard against stress-induced gastric ulcers

- Prophylactic anticoagulant after monitoring the coagulation profile

Data on heart rate, blood pressure, and bicarbonate level were collected from patients' files on the second day after admission to the intensive care unit.

Patients in the case group were subjected to the following:Initial assessment and stabilization.

• SMOFlipid 20% (purchased from Fresenius Kabi Canada Ltd.) was administered as a bolus dose of 1.5 ml/kg over 1 minute followed by continuous infusion. The bolus dose was repeated every 5 minutes up to a total dose of 3 mL/kg. A maximum of two repeated doses were given until adequate circulation was restored. The bolus dose was followed by a continuous infusion of 0.25 ml/kg/min for 30 to 60 minutes. This rate was increased to 0.5 ml/kg/min in some cases if blood pressure decreased or when the clinical situation began to worsen. A total dose of 10 ml/kg was given [11].

• Close monitoring of signs of an allergic reaction during administration was performed.

• Heart rate, blood pressure, bicarbonate, serum triglycerides, liver function tests including serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) and bilirubin were measured on admission to the intensive care unit and on the second day after administration of SMOFlipid.

• Patients were monitored noninvasively using electrocardiogram (ECG) leads attached to monitors reflecting heart rate (Mindray, Dragger, GE).

• Blood pressure was measured manually with a sphygmomanometer.

• Bicarbonate levels were tested using a GEM 5000 blood gas analyser.

• Serum triglycerides and liver enzymes were measured using laboratory tests.

# Ethical considerations

The Ethics Committee of the Alexandria Faculty of Medicine approved this study (IRB No: 00012098, FWA No: 00018699) and informed consent was obtained from the patients' next of kin before conducting the study.

# Statistical analysis

Data analysis was performed using the IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). The Kolmogorov-Smirnov test was applied to attest to the normality of the distribution of variables. Comparison of categorical variables was performed using the chi-square test. The Mann-Whitney test was applied to compare quantitative variables between two groups that were not normally distributed. At the same time, the Wilcoxon signed ranks test was used for comparisons between two periods for nonnormally distributed quantitative variables. Student's t-test was performed to compare the two groups for normally distributed quantitative variables. Paired t-tests were assessed for comparisons between two periods for normally distributed quantitative variables. The significance of the obtained results was judged at the 5% level.

# Results

The present study was conducted on 66 hemodynamically unstable patients with acute AIP toxicity. No significant difference was observed between the case and control groups with regard to gender and age (p=0.873 and 0.611, respectively). However, the mortality rate was significantly higher in the control group (p=0.001) (Figure 1).

Hemodynamic and acid-base status: (Table 1)

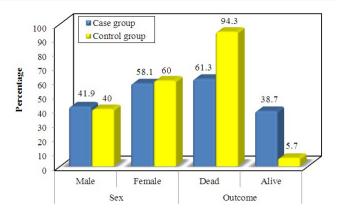
Non-significant differences were noticed between cases (before SMOFlipid infusion) and controls regarding systolic blood pressure, diastolic blood pressure and heart rate (p=0.809, 0.660 and 0.846).

There were significant differences among cases before and after SMOFlipid infusion (p=0.019 and 0.017, respectively), and between cases (after SMOFlipid infusion) and controls in terms of systolic and diastolic blood pressure (p=0.006 for both variables).

A significant increase was observed in the level of bicarbonate among cases before and after SMOFlipid infusion (p=0.006).

# Follow-up parameters of the case group

Significant increases in the levels of liver enzymes, including SGOT and SGPT, triglycerides (TGs) and bilirubin, in the cases after SMOFlipid infusion compared with before infusion (p= <0.001). (Table 2)



**Figure 1.** Comparison between the two studied groups according to gender, age and outcome (n=66)

**Table 1.** Comparison between the two studied groups according to hemodynamic and acid-base status

	Case g	Control group		
	Before infusion	After infusion	Control group	
Systolic	(n=20#)	(n=16#)	(n=25#)	
Min. – Max.	60 - 90	60 - 110	50-95	
Mean ± SD.	76 ± 11.4	90.6 ± 18.4	75.2±10.6	
Sig. bet. groups	tpp1=0.019*, tp2=0.809, tp3=0.006*			
Diastolic	(n=20#)	(n=16#)	(n=25#)	
Min. – Max.	30 - 60	30 - 80	30-60	
Mean ± SD.	45.5 ± 12.8	57.5 ± 16.1	44.0± 9.0	
Sig. bet. groups	tpp1=0.017*, tp2=0.660, tp3=0.006*			
HR	(n=31)	(n=31)	(n=35)	
Min. – Max.	98 - 167	90 - 165	95–169	
Mean ± SD.	128.2 ± 16.3	121.8 ± 17.9	127.4 ± 15.5	
Sig. bet. groups	tpp1=0.117, tp2=0.846, tp3=0.174			
HCO3	(n=31)	(n=31)	(n=35)	
Min. – Max.	3 - 14	4 – 19	4 - 14	
Mean ± SD.	8.6 ± 3.5	10.3 ± 5.2	8.9 ± 3.1	
Sig. bet. groups	Zpp1=0.006*, Up2=0.737, Up3=0.498			

tp: Paired t-test, Zp: Wilcoxon signed ranks test, t: Student's t-test, U: Mann-Whitney test p1: p-value comparing before and after, p2: p-value comparing before and control p3: p-value comparing after and control, \*: Statistically significant at  $p \le 0.05$  #: Missed cases with unrecorded blood pressure

**Table 2.** Comparison between before and after infusion of ILE

 in the case group according to follow-up parameters (n=31)

	Before infusion	After infusion	Test of sig.	р
SGOT				
Min. – Max.	30 - 98	41 – 789	7 4 7 4 4 *	<0.001*
Mean ± SD.	49.4 ± 18.8	151.5 ± 180	Z=4.744*	
SGPT				
Min. – Max.	37 - 114	35 - 854	Z=4.754*	<0.001*
Mean ± SD.	55.5 ± 19.5	171.5 ± 173.1	Z=4.734	
TG				
Min. – Max.	110 – 220	120 - 230	. 7.005*	<0.001*
Mean ± SD.	140.9 ± 26.6	152.9 ± 29.7	t=7.895*	
Bilirubin				
Min. – Max.	0.6 - 1.8	0.9 - 5	Z=4.868*	<0.001*
Mean ± SD.	1 ± 0.3	1.8 ± 0.9	Z=4.808	

t: Paired t-test, Z: Wilcoxon signed ranks test, p: p-value comparing before and after infusion, \*: Statistically significant at  $p\le 0.05$ 

Relationship between ILE infusion and the outcome

Systolic blood pressure, diastolic blood pressure and bicarbonate levels were significantly higher survivors than in cases who died (p=0.003, 0.010 and <0.001). At the same time, the heart rate was significantly lower in the survivors than in the cases who died (p=0.043).

SMOFlipid infusion caused significant decreases in liver enzymes (SGOT and SGPT) and bilirubin in survivors compared with cases who died (p=<0.001, 0.001 and 0.004). Regarding the level of triglycerides, non-significant differences were noted in survivors compared to the cases who died (p=0.269) (Table 3).

**Table 3.** Changes due to ILE infusion in the case group in relation to the outcome (n=31)

parameter	Outcomes of tl	U	р	
	Dead	Alive		
Systolic				
Min. – Max.	-20.0 - 0.0	0.0 - 30.0	1.0*	0.003*
Mean ± SD.	-7.5 ± 9.6	16.4 ± 10.3		
Diastolic				
Min. – Max.	-20.0 - 0.0	0.0 - 30.0	3.0*	0.010*
Mean ± SD.	-5.0 ± 0.10	13.6 ± 9.2	5.0"	
HR				
Min. – Max.	-48.0 - 32.0	-47.0 - 4.0	64.0*	0.043*
Mean ± SD.	-1.1 ± 24.3	-14.9 ± 15.6	64.0	
HCO3				
Min. – Max.	-3.0 - 5.0	2.0 - 6.0	19.0*	<0.001*
Mean ± SD.	0.0 ± 2.4	4.2 ± 1.4	19.0	
SGOT				
Min. – Max.	10.0 - 699.0	-12.0 - 47.0	28.0*	<0.001*
Mean ± SD.	153.9 ± 193.4	20.1 ± 18.5		
SGPT				
Min. – Max.	13.0 - 740.0	-15.0 - 95.0	34.5*	0.001*
Mean ± SD.	169.2 ± 182.0	31.8 ± 28.8		
TG				
Min. – Max.	2.0 - 30.0	-4.0 - 30.0	86.0	0.269
Mean ± SD.	13.4 ± 7.9	9.9 ± 9.4		
Bilirubin				
Min. – Max.	0.2 - 3.9	0.2 – 0.7	45.5	0.004*
Mean ± SD.	1.1 ± 0.9	0.4 ± 0.2		

U: Mann-Whitney test, \*: Statistically significant at p ≤ 0.05

# Discussion

Pesticide toxicity is a very common and challenging health problem as a method of self-poisoning, especially in developing countries, where its sale is not restricted. In Egypt, in recent years, toxicity due to the ingestion of rice tablets or aluminum phosphide (AIP) has become the most common method of suicide, especially in rural areas [12].

The exact mechanism by which AIP exerts its effects has not yet been known. Therefore, no specific antidote is available, and the treatment of such cases depends mainly on supportive measures. [13]

Many previous studies have attempted to develop new modalities for the treatment of cases of acute AIP toxicity, which depended mainly on decreasing acidity and avoiding contact

with aqueous medium, such as using paraffin oil or coconut oil for gastric lavage instead of potassium permanganate. In addition, the use of coenzyme Q10 (CoQ10), which can recover cardiac functions by increasing the activity of cytochrome C oxidase, was also proposed for the treatment of acute AIP toxicity. These measures consecutively restore mitochondrial functions and ATP production and improve the contractility of the heart. Furthermore, CoQ10 can aid in improving the multiorgan dysfunction caused by acute AIP poisoning [14-16]. Another study carried out by Goharbari et al. (2018) concluded that oral liothyronine was effective in improving the hemodynamic status of patients with acute AIP poisoning [17]. However, when the patients continued to be hemodynamically unstable or developed cardiopulmonary arrest despite all the supportive measures employed, admission to the intensive care unit was mandatory. Infusion of intravenous lipid emulsion (ILE) has been proposed by many previous studies as a method that can generate a lipid sink, which attracts the lipophilic toxic agent from tissues into the lipid phase, where it becomes ineffective [10]. Although intravenous lipid emulsion (ILE) has been used primarily in the treatment of toxicity by local anaesthetics, its use has extended to include other lipophilic poisons.

The aim of the present study was to examine the efficacy of intravenous lipid emulsion as a treatment measure for unstable cases of acute aluminium phosphide poisoning admitted to the Intensive Care Unit of Alexandria Main University Hospital, Egypt.

The type of intravenous lipid emulsion used in the current study is SMOFlipid, a third-generation lipid emulsion that is considered ideal, with a well-adjusted and more natural composition of fatty acids. Moreover, SMOFlipid is metabolized approximately 40% faster than soybean oil emulsions [18].

The dose regimen of SMOFlipid infusion used in the current study, with a total dose of 10 mL/kg, was chosen according to the American Society of Regional Anaesthesia and Pain Medicine (ASRA) and the European Resuscitation Council (ERC) Guidelines on Resuscitation. This dose was considered safe and was recommended to avoid possible side effects of ILE, which are likely to occur after a dose of 60 ml/kg and mainly affect the liver and lung tissues [19].

The mortality rate in the present study, which is the primary outcome, was calculated in relation to the number of unstable patients with acute AIP poisoning admitted to the intensive care unit only, and not to the total number of cases admitted to the poison centre. There was a significant decrease in mortality in the case group (61.3%) compared with the control group (94.3%), which demonstrated that ILE infusion helped improve the outcomes of the cases. This mortality rate was lower than in many previous studies [15,20,21].

The current work showed significant increases in systolic and diastolic blood pressures in the case group after infusion compared to before infusion of ILE and in the control group. These improvements were associated with a significant decrease in the administered amount of vasoactive drugs, mainly noradrenaline, infused in the patients. At the same time, the case group, after infusion of ILE, showed a significant increase in the level of bicarbonate compared to the level before infusion, and this rise was also reflected in the pH level,

denoting improvement of the haemodynamic compromise and metabolic acidosis caused by acute AIP poisoning. In fact, cardiogenic shock is the main cause of mortality in AIP. This result coincided with the case report carried out by Baruah et al. (2015) [22], who reported positive outcomes of the two cases studied. They also reported that ILE is better than coconut oil since it eliminates the poison after its dissolution in the plasma. Regarding the follow-up investigations of the case group before and after SMOFlipid infusion, the current study showed significant increases in the levels of liver enzymes (SGOT and SGPT) and bilirubin. These differences could be explained by the hemodynamic instability of the patients leading to ischaemic hepatitis [23]. Once blood pressure was improved, we detected improvements in liver enzymes and bilirubin levels on daily routine follow-up examinations performed regularly. Additionally, the dose of SMOFlipid infusion applied in the current work was less than needed to induce the side effects of intravenous lipid emulsion [19].

# Conclusion

The present study concluded that intravenous lipid emulsion (SMOFlipid) helped significantly decrease the mortality rate of hemodynamically unstable acute AIP poisoning cases. In addition, hemodynamic and acid-base status improved after infusion.

## Recommendations

Further studies are recommended on larger numbers of acute AIP poisoned patients (both adults and children). At the same time, future studies are proposed to study the early initiation of ILE therapy in acute AIP poisoning.

## Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

#### Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

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#### **Conflict of interest**

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

### References

1. Farzaneh E, Ghobadi H, Akbarifard M, Nakhaee S, Amirabadizadeh A, Akhavanakbari G, et al. Prognostic factors in acute Aluminium Phosphide poisoning: A Risk-Prediction Nomogram Approach. Basic Clin Pharmacol Toxicol. 2018; 1-9.

2. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arch Hyg Rada Toksikol. 2012; 63(1): 61–73.

3. Abdollahi M, Mehrpour O. Aluminum Phosphide. In: Wexler P, editors. Encyclopedia of Toxicology. 3rd edition. Oxford, UK: Academic Press; 2014. p.164-6.

4. Mehra A, Sharma N. ECMO: A ray of hope for young suicide victims with acute aluminum phosphide poisoning and shock. Indian Heart J. 2016; 68(3): 256–7.

5. Bajpai SR. Aluminium phosphide poisoning: management and prevention. J Indian Acad Forensic Med. 2010; 32:352–34.

6. Vanek VW, Seidner DL, Allen P, Bistrian B, Collier S, Gura K, et al. A.S.P.E.N. position paper: Clinical role for alternative intravenous fat emulsions. Nutr Clin Pract. 2012;27(2):150-92.

7. Adolph M, Heller AR, Koch T, Koletzko B, Kreymann KG, Krohn K, et al. Lipid emulsions - Guidelines on Parenteral Nutrition, Chapter 6. Ger Med Sci. 2009; 7: Doc22.

8. Zausig YA, Zink W, Graf BM. Lipophilicity of local anesthetics and success of

lipid emulsion therapy. Crit Care Med. 2012; 40(1):359-60.

9. Goulet O, Antébi H, Wolf C, Talbotec C, Alcindor LG, Corriol O, et al. A new intravenous fat emulsion containing soybean oil, medium-chain triglycerides, olive oil, and fish oil: a single-center, double-blind randomized study on efficacy and safety in pediatric patients receiving home parenteral nutrition. JPEN J Parenter Enteral Nutr. 2010; 34(5):485-95.

10. Rothschild L, Bern S, Oswald S, Weinberg G. Intravenous lipid emulsion in clinical toxicology. Scand J Trauma Resusc Emerg Med. 2010; 18:51-8.

11. Neal JM, Bernards CM, Butterworth JF 4th, Di Gregorio G, Drasner K, Hejtmanek MR, et al. ASRA practice advisory on local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010; 35(2):152-61.

12. Hussein HAM, Elsawaf HEM. Suicidal poisoning in Alexandria, Egypt-An updated statement. Ann Clin Anal Med. 2021;12(8):888-894

13. Pannu AK, Bhalla A. A Simple Tool Predicts Mortality in Aluminum Phosphide Self-poisoning. Indian J Crit Care Med. 2020; 24(9): 755–6.

14. Singh Y, Joshi SC, Satyawali V, Gupta A. Acute aluminium phosphide poisoning, what is new? Egypt J Intern Med. 2014; 26: 99–103.

15. Darwish RT, Sobh ZK, Hamouda EH, Saleh EM. The efficacy of Coenzyme Q10 and liquid paraffin oil in the management of acute aluminum phosphide poisoning. Toxicol Res. 2020; 9(4): 444–53.

16. Marashi SM, Majidi M, Mehran Sadeghian M, Jafarzadeh M, Mohammadi S, Nasri-Nasrabadi Z. Protective role of coenzyme Q10 as a means of alleviating the toxicity of aluminum phosphide: An evidence-based review. Tzu Chi Medical Journal. 2015; 27(1):7-9.

17. Goharbari MH, Taghaddosinejad F, Arefi M, Sharifzadeh M, Mojtahedzadeh M, Nikfar S, et al. Therapeutic effects of oral liothyronine on aluminum phosphide poisoning as an adjuvant therapy: A clinical trial. Hum Exp Toxicol. 2018; 37(2) 107–17.

18. Murilloa AZ, Jáuregui EP, Armendáriz JE. Parenteral nutrition-associated liver disease and lipid emulsions. Endocrinol Nutr. 2015; 62(6):285-9.

19. Tampakis K, Vogiatzakis N, Kontogiannis C, Spartalis M, Ntalianis A, Spartalis E, et al. Intravenous lipid emulsion as an antidote in clinical toxicology: a systematic review. Eur Rev Med Pharmacol Sci. 2020; 24(12):7138-48.

20. Saleh AA, Makhlof MG. Outcome of toxicity and mortality predictors of aluminum phosphide poisoning in Fayoum governorate, Egypt. Zagazig J Forensic Med. @ Toxicology. 2018; 16(2):40-52.

21. Sheta AA, El-Banna AS, Elmeguid RA, Mohamed HE, Gad NH. A study of the predictive factors of mortality in acute poisoning with aluminum phosphide with special reference to echocardiography and SOFA score. Environ Sci Pollut Res Int. 2019; 26(32):33135-45.

22. Baruah U, Sahni A, Sachdeva HC. Successful management of aluminium phosphide poisoning using intravenous lipid emulsion: Report of two cases. Indian J Crit Care Med. 2015; 19(12):735-8.

23. Ciobanu AO, Gherasim L. Ischemic Hepatitis - Intercorrelated Pathology. Maedica (Bucur). 2018; 13(1):5-11.

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